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ABSTRACT

This journal issue focuses on problems caused by vitamin A deficiency and its effects on vision. Although these conditions are quite serious and widespread in some areas of the world, they can easily be treated by inexpensive means. The report begins with a discussion of xerophthalmia, the most frequent cause of blindness in preschool-age children in most developing countries. Subsequent sections of the report explore (1) the history of vitamin A deficiency among human beings; (2) terminology and units of measurement; (3) metabolism; (4) vitamin A requirements and recommended intakes; (5) dietary sources of vitamin A; (6) the epidemiology of vitamin A deficiency and xerophthalmia; (7) clinical signs of vitamin A deficiency; (8) indicators of nutritional status with respect to vitamin A; (9) criteria for determining the extent of xerophthalmia and of vitamin A deficiency, from a public health standpoint; (10) the geographic distribution and prevalence of xerophthalmia; (11) treatment; and (12) prevention. Appended material describes the anatomy of the human eye. (RH)

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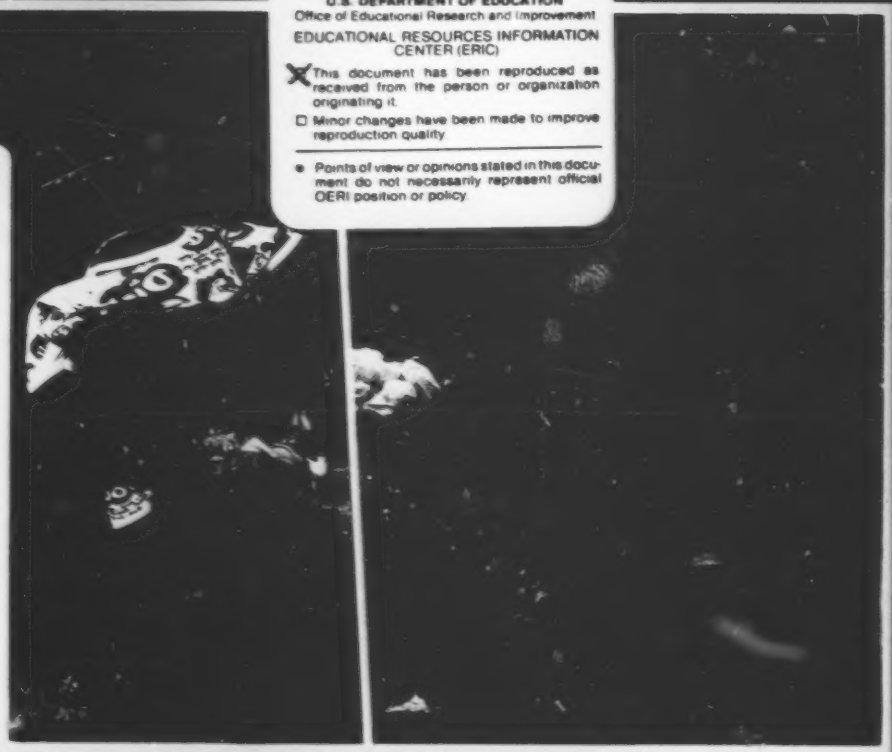

1986 N° 165 XEROPHTHALMIA AND BLINDNESS OF NUTRITIONAL ORIGIN

in the TROPICS

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The International Children's Centre was created by the French government in 1949, on the initiative of Professor Robert Debré in particular, following negotiations between France and the United Nations. Its purpose was to furnish those international and national agencies dealing specifically with child care with training facilities and educational and informational tools in the field of child health and development, viewing children within their family and surroundings.

ICC soon turned essentially toward Third World children and devoted its activities to the training and education of personnel with social, educational and administrative responsibilities as well as medical and paramedical workers. The desire for greater efficiency has led it to work increasingly with trainers and to concentrate its efforts on the methodological and educational aspects of mother and child care programmes.

ICC is also engaged in an attempt to further study — and — action on some aspects of the life and health of children and their family, so as to contribute to practical improvement, particularly in the fields of growth, nutrition, planned parenthood, the control of transmissible and nutritional diseases, preschool and school education, the needs of disabled and underprivileged children, etc.

Over this period of more than 30 years, a large amount of documents on children and adolescents, mostly from the developing countries, has been accumulated. This international documentation has been classified and sorted out, and has been computerized since 1983 : a bibliographic data base (BIRD : "Base d'Informations Robert Debré") may be consulted anywhere in the world, through international communications networks. ICC also publishes periodicals, educational documents and specialized bibliographic bulletins.

As for its legal status, the International Children's Centre is a foundation under french law of recognized public utility, administered by an executive board with broad international membership.

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EDITORIAL

This issue is devoted to the problems raised by vitamin A deficiency and its effects on vision. This is not the only domain in which vitamin A plays a role: it also seems to participate in the controlling of cell differentiation, and may play a role in checking or preventing the development of epithelial cancers. This is an extremely complex problem, and research results are not all in agreement for the moment. It is therefore extremely important that we keep informed of the findings published in the coming years.

The prevention of ocular disorders caused by vitamin A deficiency is an integral part of public health nutrition problems, the most serious of which have been discussed in this journal.

Issue 138-139-140 dealt with how to feed young children, while the composition, conservation and transformation of food was presented in issue 147-148, and the methodology of the ongoing evaluation of protein-energy nutritional status was described in issue 149-150. The various deficiencies other than those involving proteins and calories and which present a serious risk for health are gradually being discussed: nutritional anaemia (133), vitamin A deficiency, and endemic goiter, which will be the object of a forth-coming issue in 1987. Those groups suffering from an excessive weight load should not be overlooked. They exist throughout the world, in varying numbers (issue 151: the epidemiology of obesity).

Nutritional status is an overall problem, which should be approached globally within primary health care programmes. Appropriate preventive action may only be taken with some chance of success if the evolution of the nutritional status of at-risk families or groups is known. The early collection of data providing signs of the deterioration of nutritional status makes it possible to take action on an individual and collective level before the situation becomes catastrophic.

Protein and energy malnutrition weigh more heavily on the epidemiological scene than do vitamin or mineral deficiencies, but deficiency in iron, iodine or vitamin A remains a most serious problem in some areas, and it is all the more important that they be recognized and eliminated, since the lack of vitamin A, for example, may easily be remedied, and treated by inexpensive means.

by Doctor Anne-Marie MASSE-RAIMBAULT

XEROPHTHALMIA AND BLINDNESS OF NUTRITIONAL ORIGIN IN THE THIRD WORLD

by E.M. DEMAEYER

Vitamin A deficiency mostly affects small children; in severe forms, it results in blindness and death. Recent studies also seem to indicate that even mild vitamin deficiency causes an increase in morbidity and mortality due to respiratory and gastrointestinal infections. An estimated 500,000 children aged 6 months to 6 years old become blind each year, owing to vitamin A deficiency. One hundred and fifty thousand survive, to join the ranks of those who became blind during the previous years. Furthermore, 6 to 7 million children suffer from vitamin A deficiency.

This situation is all the more tragic since we are now able to treat and prevent this deficiency at a very moderate cost.

In May 1984, the World Health Assembly decided that the time had come to take action in this direction, and requested that the Secretariat of the organization prepare an international control programme, in close collaboration with the Food and Agricultural Organization (FAO) and the United Nations International Children's Emergency Fund (UNICEF). Implementation of this programme began in late 1985.

1. INTRODUCTION. SOME HISTORY

In human beings, vitamin A deficiency mostly affects the eyes. It causes a disordered physiological functioning of the rods and cones in the retina, as well as structural epithelial damage to the conjunctiva and the cornea. Those parts which are exposed to light are more severely damaged. The specific character of cutaneous hyperkeratoses and dyskeratoses is questionable, although cutaneous damage has been described during experimental deficiencies. Respiratory and intestinal disorders (diarrhoea) as well as anaemia and retarded growth have often been observed in children with vitamin A deficiency, as well as lowered defense mechanisms against infectious diseases. Furthermore, the senses of smell, taste and colour vision and of equilibrium have been reported to be disturbed during experimentally produced deficiencies.

The term "xerophthalmia" englobes all of the ocular signs of vitamin A deficiency in human beings, ranging from night blindness to keratomalacia. The latter leads to necrosis of the cornea and possible decomposition and liquefaction of the eye-ball (1,2).

Xerophthalmia is the most frequent cause of blindness in preschool-age children in most developing countries. Blindness caused by accidents only comes second, whereas it is first in the industrialized countries. Loss of vision (total or partial blindness) caused by xerophthalmia is not only a source of suffering for the young children afflicted with it; in addition, these individuals constitute a heavy social and financial burden for the community.

Xerophthalmia has been known since the times of the Pharaons. The oldest medical text known in the Western world, the **Ebers papyrus** (1600 B.C.), prescribes the eating of liver for those who suffer from it. Celsus (25 BC-50 AD) seems to have been the first to use the term xerophthalmia to describe this ailment, in his **De medicina,** which was, moreover, the first medical book to be printed during the Renaissance (1478).

Night blindness was quite frequent in Europe during the Middle Ages, and the French physician Jacques Guillemeau gave a good description of it in 1585. Much later, in 1863, another French physician, Bitot, described conjunctival and corneal xerosis, as well as the conjunctival spots which were then named after him. It is probable, however, that Bubbenet was the first (in 1860) to describe the various xerophthalmic lesions in imprisoned soldiers during the Crimean war. The nutritional origin of xerophthalmia was probably first acknowledged by Hicks, as he observed soldiers in the Confederate army during the American civil war, in 1867. Severe forms of this ailment were still observed at the beginning of the present century in Europe, in small children suffering from severe, general manutrition. The ailment has gradually disappeared on this continent, however, thanks to its socio-economic development and the improvement of medical care. By the first world war it was practically nonexistent. Minor epidemics, marked by night blindness, did occur in Denmark in 1919 and in Vienna in 1945, at times when the civilian population was suffering from a serious shortage of food.

The persistence of the ailment in Asia was known, but its extent and severity had not been studied. In 1964, then, WHO undertook a survey of its extent and geographic distribution (3). It soon became evident that the ailment was highly prevalent in Asia, causing many cases of blindness. Central America was not spared; cases of blindness were discovered there as well.

Although the nutritional origin of the disease had been suspected by 1867, it was only completely established in the 1920's. Vitamin A was isolated and its structure determined in 1930. It was crystallized in 1942 and sythesized for the first time in 1947.

2. TERMINOLOGY. UNITS OF MEASUREMENT

The term Vitamin A covers all of the compounds with the biological activity of retinol. These are, mainly, dihydroretinol, retinal and the alpha, beta and gamma carotenoids.

The concentration of preformed vitamin A in a food, a meal or a diet is generally expressed in microgrammes (μg) of retinol, regardless of whether the latter is present as free retinol or, more usually, as an ester of retinol. The amount of beta-carotene (provitamin A) is expressed in μg as well. In order to simplify the calculation of the overall vitamin content of a food product (retinol plus carotenoids), the concept of the retinol-equivalent has been conventionally adopted. It is defined as follows:

1 μg of retinol = 1 μg of retinol-equivalent (RE)
1 μg of beta-carotene of dietary origin = 0.167 μg of retinol-equivalent (RE)
and
1 μg of other carotenoids of dietary origin with biologically active vitamin A = 0.084 μg of retinol-equivalent (RE).

The international unit (IU) of vitamin A is equal to 0.3 μg of retinol.

As for the biological activity of the carotenoids, beta-carotene, and probably the other carotenoids as well, are more easily absorbed in an oily solution than when they are taken from plants. Their absorption and use by the body are inversely proportional to the amount of carotenoids present in a meal. A state of deficiency favours the absorption and use of carotenoids. When calculating the amount of biologically active vitamin in a diet, the conversion factors of beta-carotene are now considered to be approximately as follows (see table 1).

Table 1. Biological activity of beta-carotene

Beta-carotene content of meal (μg)	Conversion factor	Amount of beta-carotene equivalent to 1 μg of retinol
under 1000	0.25	4
between 1000 and 4000	0.167(1)	6
over 4000	0.10	10

(1) When information is lacking, the factor 0.167 is usually applied.

In order to facilitate calculation, it is generally considered that the amount of retinol-equivalents in a diet is equal to the sum of the μg of retinol present in the form of preformed

vitamin A, plus the amount of beta-carotene (in μg) divided by 6 and of other carotenoids (in μg) divided by 12 (see equations below):

$$\mu g \text{ retinol} + \frac{\mu g \text{ beta-carotene}}{6} = \text{RE}$$

If other carotenoids with the biological activity of vitamin A are present:

$$\mu g \text{ retinol} + \frac{\mu g \text{ beta-carotene} + \mu g \text{ other carotenoids}}{6 \qquad\qquad 12} = \text{RE}$$

3. METABOLISM

The vitamin A contained in food is present in the form of retinol, or more generally of esters of retinol, or else in the form of a precursor carotenoid, beta-carotene, which is metabolized, in the presence of an enzyme, deoxygenase, present in the intestinal epithelium and in other tissues, to form retinol. Sixty percent of the vitamin A absorbed is stored in the liver, mostly in the form of retinyl palmitate, and approximately 40% is metabolized rapidly and excreted in the faeces and urine. In a well-nourished person, the hepatic reserve represents over 90% of the vitamin A present in the body. Following hydrolysis, the esters of retinyl are bound to a specific protein, the retinol-binding protein (RBP), produced by the liver, and transported by it, in the bloodstream, to the receptor tissues. A zinc deficiency may interfere with the metabolism of vitamin A by preventing the mobilization of the hepatic reserves of vitamin A, or by interfering with the process of oxidation-reduction of vitamin A in the peripheral tissues. In the retina, for instance, the conversion of retinol to retinal and vice versa is catalyzed by a metallo enzyme containing zinc. Vitamin A is required to maintain the physical integrity of the cornea and the mucociliary functioning of the epithelial tissues. In the retina, retinol is the precursor of 11-cis retinaldehyde, which constitutes the chromophore of the visual pigments.

The serum concentration of vitamin A is relatively constant, and independent of the hepatic reserves, provided the latter do not fall below a certain threshold. A serum concentration far below normal (under 20 μg/dl) is an indication that the homeostatic control mechanisms are no longer adequate. An extremely low concentration, under 10 μg/dl, indicates the exhaustion or quasi-exhaustion of the hepatic reserves.

4. VITAMIN A REQUIREMENTS AND RECOMMENDED INTAKES

Studies made on volunteering human adults have made possible the calculation of the amount of vitamin A required for the prevention of signs of vitamin A deficiency (baseline requirements), and of the amount required for the constitution of sufficient reserves, so that the individual may cope with the hazards of everyday life. Requirements for infants were calculated on the basis of the amount furnished by the breast milk of well-nourished mothers. Requirements for children and adolescents were calculated by the interpolation

of the figures for infants and adults. During pregnancy the requirement is greater, owing to foetal growth and to the mother's need to constitute a hepatic reserve. A supplement of 100 μg of retinol/day is considered sufficient to cover foetal needs, and to enable the mother to increase her reserves. During breast-feeding, the retinol that passes into the breast milk must be replaced, thus requiring a supplement of up to 350 μg of retinol/day. Requirements are presented in table 2.

Table 2. Estimated vitamin A requirements
(in μg of retinol-equivalent/kg/day)

Age	Baseline requirements	Safety requirements
0-3 months	40	75
3-6 months	25	50
6-12 months	20	35
1-6 years	15	25
6-10 years	12	20
10-12 years	9	14
12-15 years	8	12
15-18 years	6	10
over 18 years	5	9.3

The following daily intakes have been recommended for each age group and sex, on the basis of these requirements, and taking body weight into consideration (table 3).

Table 3. Recommended daily intake of vitamin A, in μg of retinol-equivalent

Age/sex	Recommended intakes in μg/day
0-3 months	370
3-6 months	350
6-12 months	320
1-6 years	390
6-10 years	500
10-12 years	500
12-15 years	580
Boys 15-18 years	640
Girls 15-18 years	550
Adult men	600
Adult women	500
Pregnant women	600
Lactating women	850

5. DIETARY SOURCES OF VITAMIN A

Vitamin A intake is high in those regions where animal products, fruits and vegetables are abundant and eaten regularly. The best sources of preformed vitamin A - retinol - are breast milk, cow's milk, butter, liver and some fish liver oils. However, in most developing countries beta-carotene is the main source of vitamin A. It is mostly present in fruits and vegetables. Some of these are particularly rich, especially yellow or red vegetables (carrots, pumpkin, but not tomatoes), dark green leaves such as spinach and manioc or amaranth leaves, yellow fruit such as mangoes (not all varieties) and papayas, but not citrus fruit (oranges or lemons). Unrefined red palm oil is also particularly rich in beta-carotene. Some grains, such as yellow maize, and tubers such as sweet potatoes, also contain notable amounts of beta-carotene. Seasonal variations, or the lack of fruits and vegetables in the diet of small children, may considerably limit the intake of carotenoids.

According to the FAO tables of food supply, the food supply of preformed vitamin A ranges from 300 to 1000 μg per person and per day in the industrialized countries, whereas it is only 0 to 500 μg per person and per day in the developing countries. It is under 200 μg per person and per day in most of the latter. The amount of vitamin A supplied in the form of retinol and carotene ranges from 800 to 1700 μg of retinol-equivalents per person and per day in the industrialized countries, whereas it fluctuates between 100 and 1700 μg per person and per day in the developing countries. In more than half of the latter, the amount supplied is less than 600 μg per person and per day.

Table 4 shows the amount of vitamin A supplied in some countries in which xerophtalmia is a major public health problem. These figures are particularly significant when they are compared with the recommended daily intake, which ranges from 390 μg to 500 μg of RE for children aged 1 to 12 years, and is approximately 600 μg RE for adolescents and adults (see table 3).

Table 4. Food supply of vitamin A in some countries with high prevalence of xerophthalmia

Country	Retinol[1]	Carotenes[1]	Retinol-equivalents[1]
Afghanistan	142	156	298
Bangladesh	48	198	246
Burkina Faso	68	108	176
India	43	429	472
Indonesia	26	241	267
Philippines	81	421	502
Viet-Nam	59	344	403

(1) in μg per person and per day

6. EPIDEMIOLOGY OF VITAMIN A DEFICIENCY AND XEROPHTHALMIA

Vitamin A deficiency occurs when the dietary intake of vitamin A is insufficient to meet requirements. It is accompanied by a gradual exhaustion of hepatic reserves of vitamin A, leading to the occurrence of signs of xerophthalmia. These may be extremely serious, since they may result in the uni - or bilateral destruction of the cornea (keratomalacia), with the possibility of the complete disappearance of one or both eye balls. Keratomalacia is often (but not always) accompanied by signs of general malnutrition.

Xerophthalmia is most frequently encountered in pre-school children, aged 6 months to 6 years. This is not surprising, since the requirements of children of those ages are three times as high, per kg of body weight, as those of adults. In some areas, such as India, Bitot's spots are more frequently observed in school-age children, however. Night blindness is also seen in pregnant or lactating women, in some areas.

Boys seem to be affected more often than girls, and a 3/2 male: female ratio has been reported, particularly in Indonesia and India. This greater vulnerability of boys increases with age. It seems to be of endocrine origin.

Vitamin A deficiency is linked with underdevelopment, and is mostly observed in the poorest, least educated population groups. It was frequently seen in Europe during the pre-industrial era, and was present in Japan until 1930. It has disappeared quite recently in countries such as Korea and Singapore, owing to their rapid socioeconomic development. It has gone unrecognized in many places, since it affects the most marginal sectors of the population, who have no access to health care, or again, because it is linked with an infectious disease which is believed to be directly responsible for the blindness. This was the case for measles in Africa, for many years: it was believed to be the only cause of cases of blindness occurring in small children. In fact, it is often linked with protein-energy malnutrition. Both marasmic and oedematous forms are similarly afflicted.

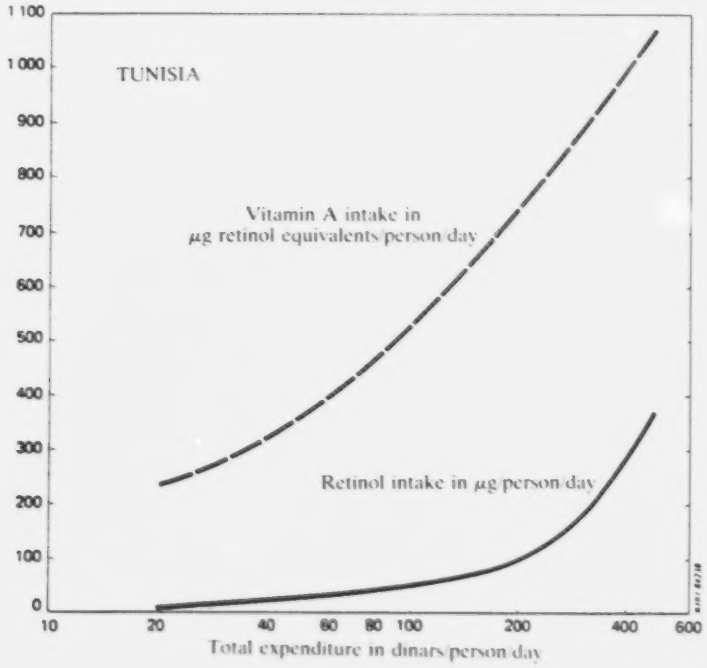
Family income and dietary vitamin A intake are closely correlated. During a diet survey performed in Tunisia (4), retinol and beta-carotene consumption was found to increase with income, although retinol intake was always behind beta-carotene (see figure 1). On a cross-cultural level, there is a similar correlation between vitamin A intake and socioeconomic development, to the point that the former is generally a good indication of the latter.

It is a widely recognized fact that childhood infectious diseases, and more particularly measles and gastrointestinal, respiratory and urinary infections, accelerate the ocurrence of xerophthalmia in children with little or no hepatic reserves of vitamin A. Epidemics of xerophthalmia have been observed following episodes of diarrhoea or of respiratory ailments. They may be seasonal, and linked with the greater frequency of certain types of infection at specific times of year (respiratory infections in winter, gastrointestinal infections during the hot season). Although many infectious diseases bring about a loss of appetite, with an attendant drop in the dietary intake of vitamin A, this phenomenon is particularly serious in measles. There is a highly significant drop in the serum vitamin A concentration, while metabolism of the vitamin increases, resulting in its increased use and excretion. Infectious diseases usually affect the intestinal absorption of vitamin A. The

serum concentration of vitamin A decreases during many infections, but returns to its prior level shortly afterward. This seems to indicate that the releasing of vitamin A from the hepatic reserves and its transportation in the bloods tream may be directly affected by infection.

Fig. 1

RETINOL AND VITAMIN A INTAKE (RETINOL AND BETA-CAROTENE) IN RELATION TO FAMILY INCOME IN TUNISIA



The fact that measles favours the occurrence of xerophthalmia may be explained by its impact on the child's nutritional status. Indeed, it is well known that epidemics of measles in Africa are usually followed, within weeks, by epidemics of protein-energy malnutrition. Although measles causes specific corneal damage, healing occurs rapidly in well-nourished children, leaving no trace. We have many reasons to believe that such damage only tends toward corneal ulceration and, in some cases, destruction, in cases with underlying vitamin A deficiency.

Furthermore, it is important to note that vitamin A plays a role in the body's defense mechanisms against infection, and that any deficiency weakens cell-mediated immunity. This close interrelation between infectious disease and vitamin A deficiency has recently been proved by Sommer et al in Indonesia. In a first study (5), they showed that children with signs of mild xerophthalmia (night blindness or Bitot's spots) ran twice as great a risk of contracting a respiratory infection and three times as great a risk of gastrointestinal infection as children with no signs of xerophthalmia and with an otherwise similar nutritional status. In a second study (6) the same authors showed that in a region where vitamin A deficiency was endemic (Sumatra), the distribution of a vitamin supplement (200,000 IU of vitamin A) twice yearly to children aged 1 to 6 years resulted in a drop of about 30% in the mortality rate, in comparison with a control group.

Blindness occurring at a very early age represents a serious handicap for the child's mental and physical development. In the Philippines, a study of school-age children who did not attend school showed that a great many of them were partially or totally blind.

The mortality rate subsequent to keratomalacia is high in children with severe malnutrition; the younger the child, the higher the rate. In all cases of corneal ulceration, only an estimated 30 to 35% of children survive, for lack of adequate treatment.

With the exception of pregnant and lactating women, adults who suffer from xerophthalmia are usually afflicted with infectious hepatitis, cirrhosis of the liver or sprue.

7. CLINICAL SIGNS

In adults, clinical signs only develop after some 12 to 18 months of a deficient diet. Signs of deficiency may develop very rapidly in small children, whose vitamin A reserves are meagre: several days or weeks may suffice.

Ocular signs constitute the only reliable clinical indication of vitamin A deficiency. Even signs representing less advanced stages, such as night blindness and Bitot's spots, should be retained, since they sound the alarm. The first clinical signs are only slightly correlated with the serum vitamin A concentration. An experienced observer can easily detect the former without any special equipment, whereas a well-equipped laboratory is required to assay the serum vitamin A concentration.

In human beings, ocular signs of vitamin A deficiency include night blindness, xerotic damage to the conjunctiva and cornea, ulceration and destruction of the cornea (keratomalacia), corneal spots and modifications of the retina. The term "xerophthalmia" should not be confused with "keratomalacia", the meaning of which is far more restricted, and which only applies to the process of destruction of the cornea and eyeball, seen exclusively in the most severe forms of vitamin A deficiency.

The existence of other causes of xenosis of the conjunctiva, Bitot's spots and night blindness should be recalled, although such cases are infrequent.

Symptomatology and classification of xerophthalmia

Night blindness (XN)

This term is preferable, since it avoids any confusion due to differences between French and English usage. Indeed, "hemeralopie" is used in French to designate any failure or imperfection of vision in dim light, whereas in English the same word, "hemeralopia", has the opposite meaning, that of day blindness, or of better vision in dim light. The same danger exists for the term nyctalopia, which means night blindness in English, while in French "nyctalopie" indicates the ability to see at night, or to distinguish objects better in dim light than in bright light. For this reason, the term "night blindness" is used here to the exclusion of all others.

When the amount of vitamin A aldehyde (retinone, retinal) supplied to the retinal rods is decreased, the adjustment of vision to dim light is disturbed. This decrease in supply may be detected long before the person complains of an absence of night vision, by the measurement of adjustment to darkness, or by electroretinography. However, both techniques are difficult to use in the environmental context in which vitamin A deficiency is generally observed; in addition, they require better cooperation by the patient than can be expected of a child. It is difficult to detect night blindness in small children. The mother may have noticed it, however, and the existence of a number of vernacular terms is an indication of its frequent occurrence in some parts of the world. Quite recently, some simple tests appropriate for use with small children have been developed, making on-the-spot diagnosis possible. One of them involves asking the mother to sit in a corner of a darkened room. An obstacle (a table or bench) is placed in the centre of the room, while the child is placed in the corner opposite from the mother's corner. When the mother calls her child, it is quite probable that he/she will run into the obstacle in case of night blindness. Although the latter is compatible with a relatively satisfactory general health status, it should be considered an important diagnostic sign, since it precedes epithelial lesions.

Conjunctival and corneal lesions

Xerosis conjunctivae may progress over a period of several weeks or months, whereas irreversible damage occurs within days of the occurence of corneal xerosis. In infants, in particular, keratomalacia may develop rapidly under closed eyelids, even when no warning signs are observed.

Xerosis conjunctivae (X1A)

The lesions characteristic of vitamin A deficiency are generally limited to the bulbar conjunctiva, but in longer lasting cases the conjunctiva of the lower eyelid and the adjacent inferior fornix (1) may become wrinkled (loose) and rough.

Xerosis may affect all of the exposed part of the bulbar conjunctiva, or be located in a small portion of it. In any case, the lesions are similar. Their characteristics are:

- Dryness (xerosis): this is judged by the dull, lusterless appearance of the bulbar conjunctiva. It has been compared to wax, or dried paint.
- Loss of the protective film: it is evident regardless of whether lacrymal secretion is present or not. The xerotic spots look like "sandbanks at low tide" when the child stops crying. They are probably produced by a gap in the preconjunctival film, caused by the xerotic process.

(1) Fornix : ocular conjunctival cul de sac.

— Loss of transparency: the lack of transmission of light through the conjunctiva results in the decreased visibility of the conjunctival vessels. When viewed through the slit lamp the conjunctiva, normally transparent (like an aquarium) and covered with blood vessels, is seen to be milky, due to presence of fine droplets. The vascular network is scarcely visible, with the exception of the largest arterioles.
— Thickening: the conjunctiva show a general tendency to thicken and become rigid.
— Wrinkling: small, more or less vertical folds appear in the conjunctiva. This is best observed by drawing the temporal part of the conjunctiva outward, with the eyeball in the extreme internal lateral position.
— Pigmentation: in dark-skinned individuals, there is a slight, diffuse, brownish colouration which should not be confused with the more gross, spotted pigmentation frequently observed in light-skinned individuals. In prolonged cases of xerosis, the inferior fornix first turns yellow, then light grey and at last, dark brown, because of the presence of chromatophores in the basal cells of the epithelium. This characteristic pigmentation responds slowly (several weeks, at the least) to treatment.

Bitot's spots (X1B). (Figures 2, 3)

Bitot's spots are generally small, soapy patches with a silvery-grey reflection. They are relatively superficial and are raised with respect to the conjunctiva. They may easily be removed by rubbing, but then reveal a rough-surfaced underlying xerotic conjunctiva.

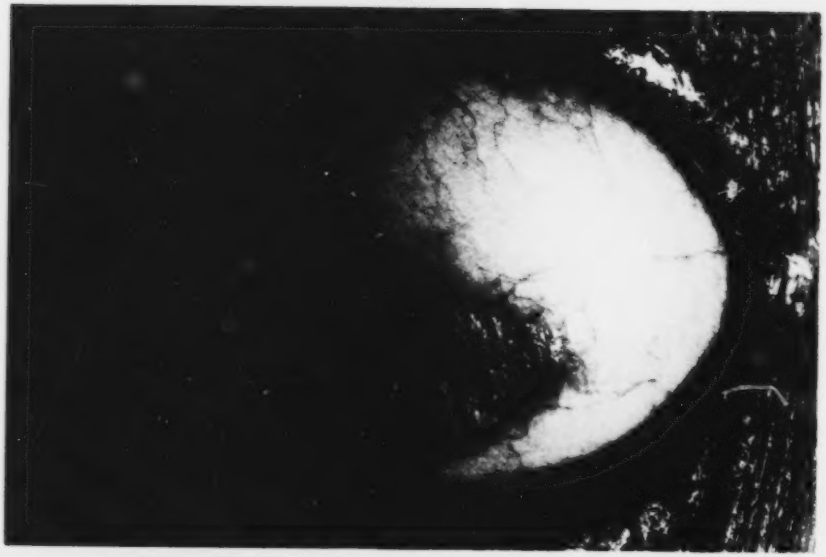
Bitot's spots are usually triangular and are always located on the bulbar conjunctiva. They are generally temporal, and only rarely located on the nasal side. They are often bilateral. This typical location is probably due to the fact that it is protected from the rubbing of the eyelids, and also sheltered by the limbus, which is slightly protuberant. However, the classical, triangular shape may vary, producing irregularly circular or oval spots, along a horizontal axis. Other shapes are seen in some rare cases,when the material composing the Bitot's spot is scattered over the conjunctiva or shows vertical lines resembling "corrugated iron". These spots are not always soapy some are cheesy of fatty in appearance. Others are not flat, and may even be quite bulky, when the material accumulated is considerable. The meaning of these variations is unknown. The spots may be black when the child's eyelids are made up with mascara.

Bitot's spots are not necessarily linked with generalized xerosis conjunctivae. When they are, the patient is generally a small child in whom night blindness may also be present. Like xerosis, these spots usually respond well to treatment. Bitot's spots alone, without xerosis conjunctivae, may occur in adolescents and adults. They may not respond to the administration of vitamin A, and are then probably not caused by a deficiency in this vitamin. In some areas such as India, Bitot's spots are more frequently observed in school-age children than in children under six, and respond well to the administration of vitamin A.

Accumulation of debris

In some cases of advanced xerosis, debris may accumulate on the surface of the bulbar conjunctiva, and may also cover the adjacent parts of the cornea. They are creamy white, shiny, not soapy and are easily removed. They may be found in the inferior fornix, at the meeting of the eyelids or on their edges, but such cases are rare. Bitot's spots are far more frequent, and their substance adheres firmly to the eye.

Fig. 2



Bitot's spot, soapy type.

Fig. 3

Bitot's spot, creamy type.

Corneal lesions

Xerosis of the cornea is generally but not necessarily consecutive to the occurrence of xerosis conjunctivae. Both corneas may be affected to differing degrees. Photophobia and inflammation are often absent, or may be quite mild. In un-complicated cases, ocular congestion is remarkably discrete.

1. Xerosis of the cornea (X2) (Figure 4) is reversible and may be generalized. It is characterized by inability of the cornea to remain moist, and therefore shiny, marked dryness (or "non-wetability") and a loss of transparency, resulting in a blurred appearance. These changes are clearly seen when the eyelids are held apart for 15 seconds. A gap in the epithelial surface may also occur. Later, cellular infiltration of the corneal stroma further reinforces the blurred appearance of the cornea, which comes to look milky and bluish, particularly in the inferior segment of its central part. A cellular exudate may also be noted in the lower part of the anterior chamber in some cases.

2. Irreversible lesions are characterized by the following signs:

— corneal ulceration affecting less than one third of the cornea (X3A). The term ulceration is imperfect, since it actually designates a loss of substance within some or all layers of the cornea. Only minor signs of reaction or inflammation occur, except in the case of concomitant secondary infection. When the ulceration takes the course of a major loss of the stroma, it may result in a descemetocele (1), and complete perforation, with prolapse of the iris. These lesions are more frequent in the lower half of the cornea; when treated they leave sequelar corneal opacity which impairs vision.

— corneal ulceration/keratomalacia affecting more than one third of the cornea (X3B) (Figure 5). This usually occurs in conjunction with severe protein-energy malnutrition combined with vitamin A deficiency, but not always. Most of the cornea is damaged, and lesions may take the form of characteristic softening (colliquative necrosis) throughout the cornea, leading to the distortion and destruction of the eyeball. The evolution is rapid: the corneal structure is disintegrated, leaving a white or dirty yellow gelatinous mass. In the absence of treatment, panophthalmia (2) may occur. Keratomalacia may develop rapidly, with no warning lesions. The extremely mild reaction of the surrounding structures is a characteristic feature of this ailment.

Scars (XS)

The sequelae which are least disabling for vision are ocular opacity and small leukomas with no involvement of the pupillar region. They are generally located on the lower segment of the central part of the cornea. In case of an iridocele (3), an adherent leukoma (4) occurs, with distortion of the pupil. Large, total or subtotal leukomas, often highly vascularized and pigmented, result in loss of vision, often limited to a single eye.

Keratomalacia may cause an anterior staphyloma (5), composed of the cicatricial vestiges of the cornea plus material from the uvea, which bulge outward under the increased intraocular pressure. When the cornea is torn instead of bulging, the ocular contents are expelled, leaving an atrophied eyeball.

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(1) Descemetocel : a herniation of descement's membrane, which borders the posterior side of the cornea.

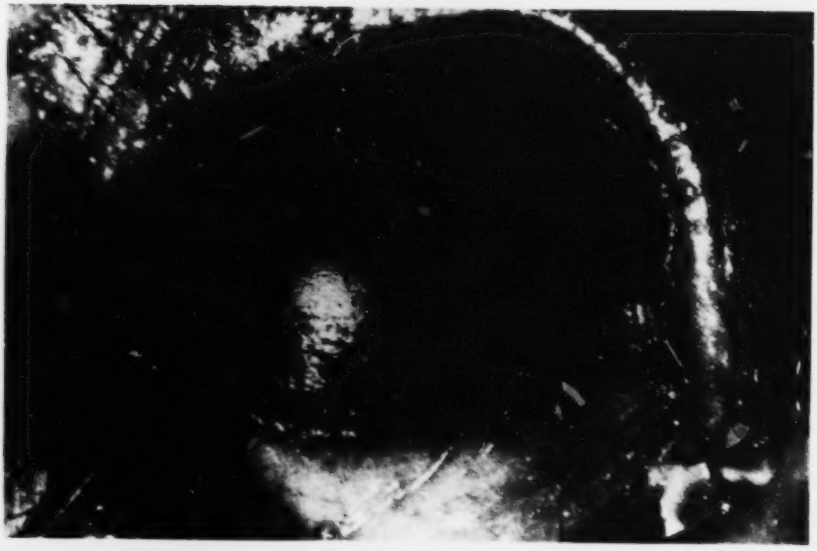
(2) Panophtalmia : purulent ocular inflamation.

(3) Iridocele : the iris trotrudes through the cornea.

(4) Lucoma adhaerens : a thick, white, scar on the cornea, enclosing a part of the iris.

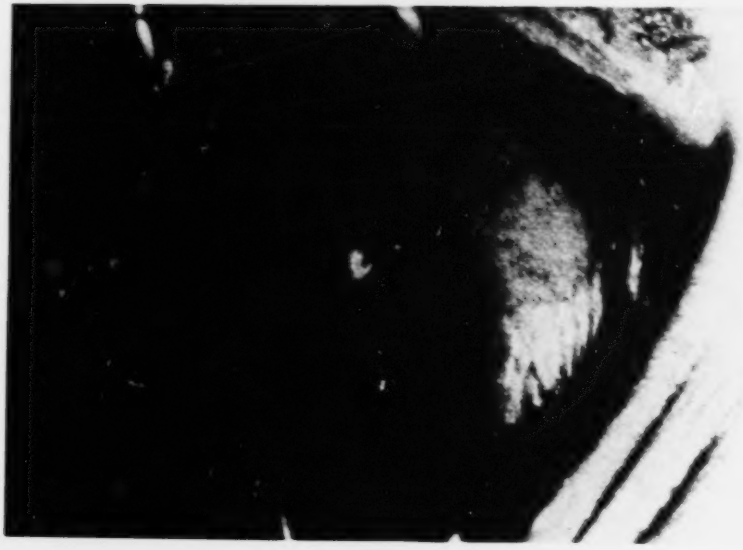
(5) Staphyloma : trotrusion of the cornea or of both cornea and sclera.

Fig. 4



Xerosis of the conjunctival and cornea. The cornea books dry and granular.

Fig. 5



Bitot's spot, and keratomalacia.

20

Fundus of the eye in xerophthalmia (XF)

Lesions of the posterior segment of the eye are described as white spots spread along the vessels at the retinal level. These spots may run together. They are generally seen around the periphery of the fundus. They probably represent a depigmentation process affecting the tissue containing the retinal pigment.

It should be noted that until the corneal xerosis phase, xerophthalmic lesions are entirely curable by proper treatment, and leave no trace. Furthermore, there is no strict chronological order in the succession of the various signs described above, and corneal xerosis may occur, for example, without any warning signs such as Bitot's spots. Such is the case in Haïti, for instance, where xerosis of the cornea is relatively frequent, while Bitot's spots are rarely seen.

8. INDICATORS OF NUTRITIONAL STATUS WITH RESPECT TO VITAMIN A

Various indicators have been used to describe nutritional status with respect to vitamin A. These include: dietary retinol and carotenoid intake; measurement of the vitamin A and carotenoid content of body fluids and tissues; detection of the various clinical signs of xerophthalmia. Each of these indicators furnishes some specific information, which has its limits as far as interpretation is concerned. When all indicators are in agreement, the diagnosis is clear. In case of severe deficiency, for instance, practically all of the indicators and particularly the clinical signs and biochemical parameters, will be closely correlated. When the deficiency is less severe, the correlation is not as good. Thus, signs of xerophthalmia may be observed in children with a serum retinol level of 10 to 15 μg/dl. In other cases, children with a serum retinol level below 10 μg/dl may not show any clinical signs of xerophthalmia. This shows how complex the situation is, when the status of any one individual must be assessed.

It is easier to evaluate the nutritional status of a population or group of individuals, since the correlations between the different indicators, and more specially between the clinical and biochemical indicators, are more clearly statistically significant, and easier to interpret.

9. CRITERIA FOR DETERMINING THE EXTENT OF XEROPHTHALMIA AND OF VITAMIN A DEFICIENCY, FROM A PUBLIC HEALTH STANDPOINT

It may be necessary to decide whether the deficiencies observed in a given population group are sufficiently serious to merit the establishment of a broad prevention programme, and if so, to define the type of programme. The following criteria have been suggested, although some are rather arbitrary, given the present state of our knowledge.

Clinical signs

— Night blindness (XN): prevalence of 1% or more.
— Bitot's spots (X1B): prevalence of 0.5% or more.
— Xerosis of the cornea, corneal ulceration, keratomalacia (X2/X3A/X3B): prevalence of 0.01% or more.
— Corneal scar (XS): prevalence of 0.05% or more.

Biochemical signs

— Serum vitamin A level under 0.35 micromoles/L. (100 mcg/l) in over 5% of the at-risk population.

Comments

— These criteria apply to at-risk groups; that is, usually to groups of preschool children (1 to 5 year-olds). Prevalence must be calculated for a representative sample of this population group.
— The presence of one clinical sign should be interpreted as an indication of a significant xerophthalmia problem. The presence of several signs further reinforces the seriousness of the situation.
— In the absence of any clinical signs, the biochemical sign constitutes an indication of an insufficient vitamin A intake and a latent state of vitamin A deficiency within the population group. It may justify the decision to take action in order to improve nutritional status, particularly with respect to vitamin A. A dietary fortification programme may be set up, for example.
— A low vitamin A level may occasionally be observed by performing liver biopsies on accidentally deceased children. A vitamin A level of less than 5 mcg/g of fresh liver tissue in at least 5% of the individuals examined, or under 20 mcg/g in at least 20% of such individuals may be regarded as an indication of vitamin A deficiency within the population group.

10. GEOGRAPHIC DISTRIBUTION AND PREVALENCE OF XEROPHTHALMIA

Xerophthalmia constitutes a major public health problem in the sahelian and subsahelian regions of Africa (Benin, Burkina Faso, northern Ghana, Mali, Mauritania, Niger, northern Nigeria and Chad) and in the eastern and southern parts of that continent (Angola, Ethiopia, Kenya, Malawi, Mozambique, Uganda, U.R. of Tanzania and Zambia); in America, in Bolivia, Brazil, El Salvador, Haïti and Mexico; in South-East Asia, in Bangladesh, Bhutan, Burma, India, Indonesia, Nepal and Sri Lanka; in the Eastern Mediterranean area, in Afghanistan, Oman and Sudan; in the Western Pacific, in Fiji, Democratic Kampuchea, the Philippines, Lao People's Democratic Republic and Viet Nam (Figure 6). This list is not exhaustive, and other countries may be added to it as more information is collected. Furthermore, vitamin A deficiency with sporadic cases of xerophthalmia is present in many developing countries. However, the number of cases is not great enough to constitute a high-priority problem (7).

Although the fragmentary nature of available data makes it difficult to accurately estimate the number of cases of xerophthalmia occurring each year, the findings provided by thorough surveys conducted in some countries clearly show the extent of the problem.

For example: it has been calculated that each year, a total of some 400,000 preschool children in Bangladesh, India, Indonesia and the Philippines show active corneal lesions resulting in partial or total blindness. The total annual number of cases of non-corneal xerophthalmia (mild, reversible forms of the disease) in these four countries is probably somewhere around 5 million for preschool-age children.

If we project these estimations on a world-wide scale, we arrive at a total of 500,000 new cases of active corneal lesions each year, and a total of 6 to 7 million cases of non-corneal xerophthalmia. In the hypothesis that 50% of those 500,000 cases of corneal lesions result in total blindness of one or both eyes, and that 60% of these children die within weeks of the occurrence of uni or bilateral blindness, 150,000 children who are blind in one or both eyes survive each year, and join the ranks of those who became blind during previous years. These estimations are very conservative, and do not take into account the enormous number of other children who suffer from a lack of vitamin A, although they do not show any signs of active xerophthalmia.

11. TREATMENT

All cases of xerophthalmia, including cases of night blindness, conjunctival and corneal xerosis should be rapidly and vigourously treated. This is particularly true for small children, since lesions may develop extremely rapidly. It is therefore advisable that all medical and paramedical workers be capable of recognizing and treating xerophthalmia and keratomalacia, and that satisfactory preparations containing vitamin A be available in all health centres, from the village health unit to the most sophisticated hospital. The following treatment protocol is suggested:

When diagnosed: 200,000 IU of an oily preparation of retinyl acetate or palmitate, administered orally. If the child is unable to swallow, vomits or suffers from profuse diarrhoea, as is often the case in severe forms of protein-energy malnutrition, it is preferable to administer 100,000 IU of an aqueous suspension of retinyl palmitate intramuscularly.

On the second day: 200,000 IU of an oily preparation of vitamin A (110 mg of retinyl palmitate or 66 mg of retinyl acetate), orally.

When discharged from hospital, or after about ten days: 200,000 IU of an oily preparation of vitamin A, orally (110 mg of retinyl palmitate or 66 mg of retinyl acetate).

Comments

The aqueous suspension of retinyl palmitate should be preferred for intramuscular administration.

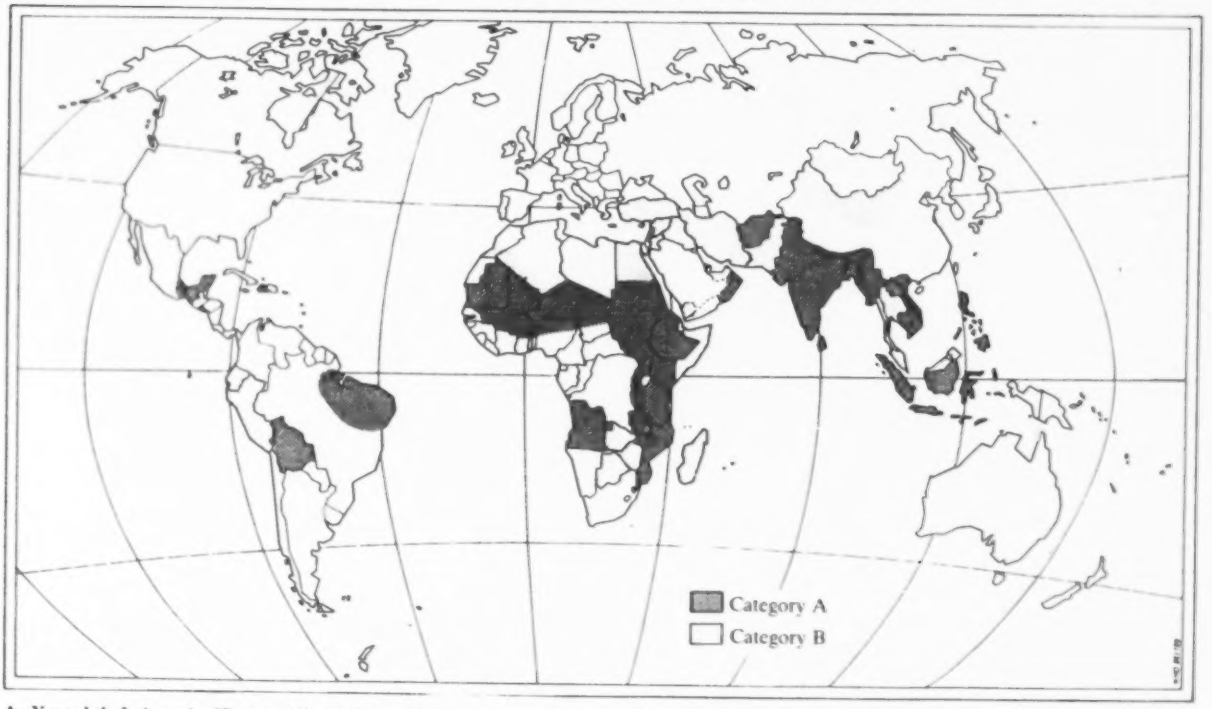
Oily solutions should not be injected, since resorption of vitamin A is extremely slow, and its action unreliable.

For oral administration, an oily solution of vitamin A is generally used.

The doses administered should be halved for infants under one year old.

Fig. 6

THE GEOGRAPHICAL DISTRIBUTION OF XEROPHTHALMIA IN 1986



A: Xerophthalmia a signifiant public health problem

B: Sporadic cases of xerophthalmia do occur

It is clear that this treatment only aims at saving vision whenever possible. Great care should be taken to treat malnutrition in general and intercurrent infections, as well. Antibiotics and a diet rich in energy and proteins should therefore be administered, in accordance with needs.

The topical application of vitamin A preparations should be avoided. These are not specially effective, and may in fact be dangerous. As a rule, the eye should not be touched. It should therefore be protected, using sterile compresses and, when needed, an antibiotic ointment.

Children showing signs of deficiency should be followed up after recovery. Massive doses of vitamin A may not be retained by the liver when the child is malnourished, and a relapse may occur if his/her diet is not permanently improved. Administration of vitamin A should be pursued for such children (200,000 IU every 4 to 6 months), along with surveillance, to make sure that their diet is sufficiently rich in vitamin A, as well as in proteins and energy.

When no medicinal preparations of vitamin A are available, it should be recalled that liver (beef, sheep, chicken or fish liver) contains between 5,000 and 50,000 mcg/100 g of vitamin A. Cod liver oil contains 30,000 mcg/100 ml and the biological action of palm oil is about half that (24,000 to 48,000 mcg/100 g). These sources of vitamin A may be quite valuable when vitamin A preparations are not available.

12. PREVENTION

1 - Nutrition education and the development of horticulture

The best preventive measure is the improvement of people's diet. This is, of course, a long-term solution, since a long period of time and a continuous, prolonged effort are required in order to bring about changes in dietary intake through the use of existing resources or the development of new ones, thanks to nutrition education.

Carotene-rich vegetables, fruits or other leafy, green plants, such as the leaves of sweet potatoes, manioc or amaranth, yellow vegetables and fruits such as carrots, mangoes and papayas exist almost everywhere. Unripe legumes are rich in both proteins and carotene.

The main problem is educating people about nutrition. It is paradoxical, for example, that the population of Java, a major producer of carotene-rich palm oil, suffers from xerophthalmia but refuses to eat red palm oil. In central Africa, people are fond of red palm oil. It is a part of the usual diet, and constitutes an excellent source of vitamin A. Nutrition education cannot solve all of the problems, however; it is vain, for instance, to recommend the inclusion of liver and meat in the diet in some countries, since most of the population is vegetarian.

Furthermore, green vegetables are often held in low esteem, and viewed as food for the poor. In addition, small children do not sem to like vegetables much, and they must often be taught to eat them. This requires time and patience, which mothers do not readily expend when they themselves do not think very highly of this type of food.

The economic and social circumstances in which people live are of course extremely important. Improved conditions immediately result in a more varied diet. This leads to an increased carotene intake, and possibly an increased retinol intake if the diet includes animal protein. The fact that vitamin A deficiency has disappeared in Western countries and in Japan, in particular, where it still took catastrophic proportions at the beginning of this century, constitutes the best proof that proper organization of preventive medicine, in conjunction with an improved standard of living, makes the complete eradication of this vitamin A deficiency possible.

II - Administration of large doses of vitamin A

The administration of single, massive doses of vitamin A to at-risk groups on specific occasions or at regular intervals may be organized rapidly, and only requires a minimum of infrastructure. This is essentially an emergency measure aimed at preventing and combating vitamin A deficiency and xerophthalmia until the problem can be definitively solved.

Infants (under 12 months old) should be given 100,000 IU orally, while 1 to 6 year-olds should recveive 200,000 IU of vitamin A. Approximately 30 to 50% of this dose will be stored by the body. The period of protection for a growing child is therefore estimated at 100 days, when vitamin A intake from other sources is nil.

The vitamin A content of the diet constitutes a major factor in determining the periodicity of administration. The usual periodicity is four to six months, but it may be shortened to 3 months when the diet is very poor (general shortage of food, or any other emergency situation), or if this makes its integration with other periodically functioning primary health care activities feasible. Because of its extremely broad coverage, this type of distribution aimed at all preschool children (6-month to 6-year olds), is also known as the "universal system".

There is another type of system, known as the medical or therapeutic system, which involves the prophylactic administration of a dose of 200,000 IU of vitamin A to all preschool children who are brought into contact with a medical unit because of a bout of diarrhoea, a case of measles or severe protein-energy malnutrition.

All prophylactic programmes should include lactating mothers. During the month following delivery, they should be given 200,000 IU of vitamin A, in order to increase the vitamin A content of their milk.

A number of general rules should be respected by all supplementation programmes:

1. The programme should be aimed at the group exposed to the greatest risk. Maximum community participation should be sought.
2. The existing infrastructure should be used whenever possible, and the programme be integrated in the permanent health activities.
3. The periodic distribution programme should be accompanied by a health and nutrition education programme.
4. A system for controlling and evaluating the programme should be established from the outset.

The dose of 200,000 IU of vitamin A is usually administered in the form of capsules with a short tab on the end. For use, the tab is cut and the contents of the capsule pressed deep into the child's throat, causing reflex swallowing. The operation is the same for children under one, but the first two drops of the oily solution are removed, thus reducting the dose to 100,000 IU. Many countries use this type of capsule. In India, however, vitamin A is administered in syrup form, using a spoon.

The universal supplementation system is presently applied in India, Bangladesh and Indonesia, in the framework of national programmes for the prevention of vitamin A deficiency. The Indonesian programme has recently been evaluated in one of the islands forming the Indonesian archipelago, Lombok, where the prevalence of xerophthalmia was particularly high (8). The capsule distribution programme began in 1977 and has continued until now. All children aged 6 months to 5 years old were given a capsule containing

200,000 IU of vitamin A twice a year. Two surveys of the prevalence of xerophthalmia were performed: one before the distribution had begun (1977), the other 6 years later (1983). During this period, the prevalence of active forms of xerophthalmia dropped from 4.62% in 1977 to 1.18% in 1983. The prevalence of Bitot's spots dropped from 1.6% in 1977 to 0.24% in 1983. Similarly, the prevalence of active corneal lesions went from 0.2% to 0.04%. The frequency of corneal scars remained stable at 0.2% (Figure 7).

On the whole, in 1983, 7.1% of children suffered from some ophthalmological disorder, including 1.2% with ocular signs of vitamin A deficiency and 5.9% with other ocular diseases (Figure 8). These results show this form of protection to be extremely effective. They also indicate that some children were not covered by the programme. Unfortunately, the same observation has been made in most programmes: coverage is not perfect, and only affects 80% of children, at the most. For a variety of rather complex reasons, it is unfortunately the most vulnerable, most marginal groups which are not covered by these programmes.

At the present time, UNICEF furnishes 200,000 IU vitamin A capsules to many countries, at a cost of approximately $US 0.02 per capsule. This does not mean that prophylaxis by the administration of vitamin A costs about $US 0.04 per child and per year, since the cost of distribution is not included in this figure. Estimation of the latter is difficult, since it varies considerably with the method used and with the extent of its integration with other health programmes.

Prophylaxis by vitamin supplements may be summarized as follows:

1. Infants under 12 months old: 100,000 IU of vitamin A in liquid or capsule form at 4-month intervals. When the standard 200,000 IU capsule is used, remove the first two drops and administer the rest of the contents.

2. Children over one: 200,000 IU of vitamin A in an oily solution, in liquid or capsule form, orally every 3 to 6 months.

3. Pregnant women and those of childbearing age: daily doses of vitamin A should not exceed 10,000 IU, owing to possible teratogenic effects.

4. During the first month of postpartum: the administration of a single oral dose of 200,000 IU of vitamin A is effective in increasing the vitamin A content of breast milk. The administration of a large dose of vitamin A at that time is not dangerous, since the probability of occurrence of another pregnancy within 30 days of delivery is practically nil.

III - Dietary fortification

Fortification of food with various nutrients has been practiced extensively for many years, and the general rules have been clearly established. In Europe and North America, for instance, margarine is commonly fortified with vitamin A (retinol and beta-carotene) for nutritional and aesthetic reasons (its yellow colour imitates the colour of butter). To be suitable for fortification with vitamin A, a food product must meet the following qualifications:

Fig. 7

PREVALENCE OF OCULAR SIGNS OF VITAMIN A DEFICIENCY, LOMBOK, INDONESIA, SEPTEMBER 1977 AND SEPTEMBER 1983

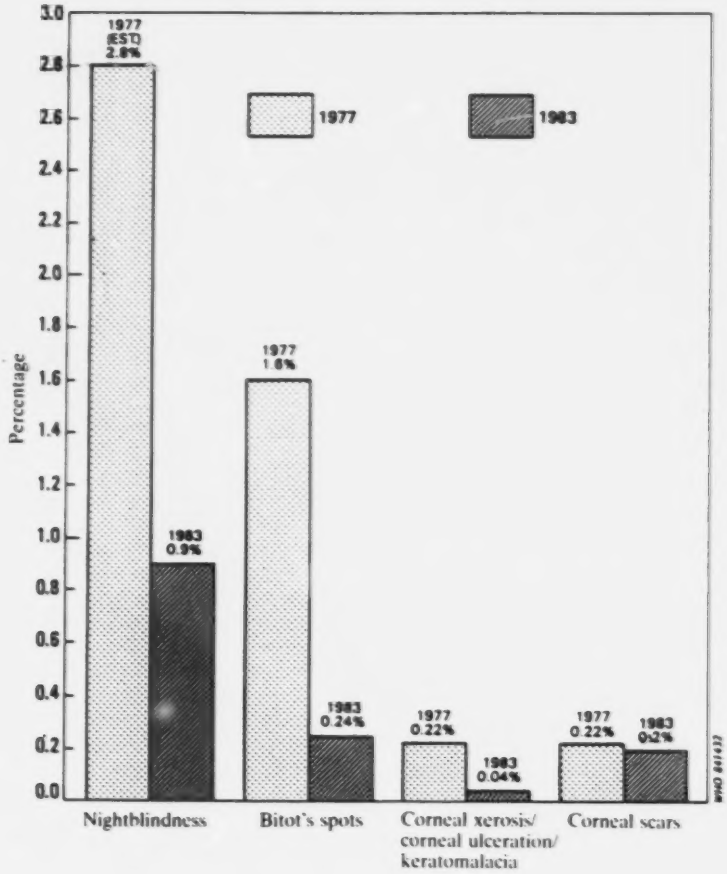
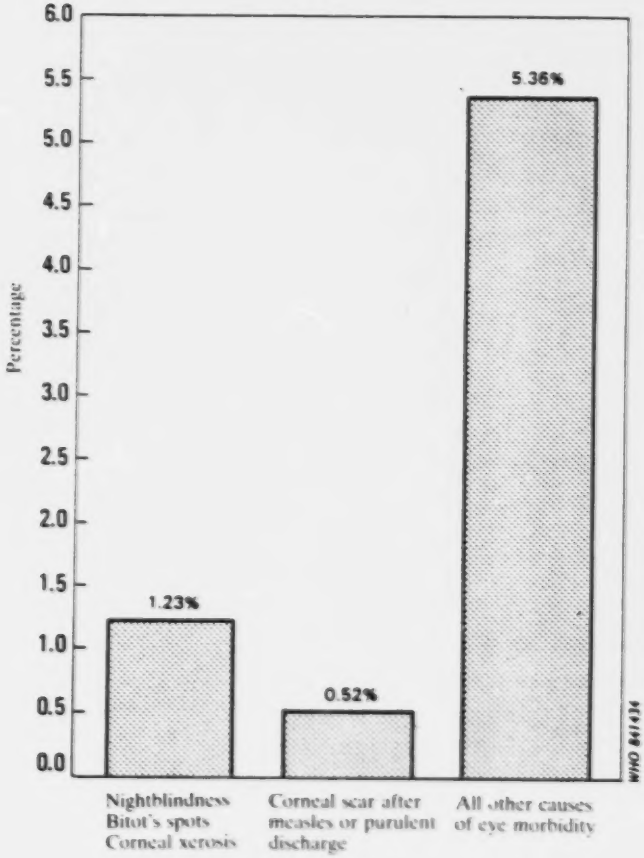


Fig. 8

PREVALENCE OF XEROPHTHALMIA, CORNEAL SCAR AND OTHER CAUSES OF EYE MORBIDITY, IN PRE-SCHOOL CHILDREN FROM LOMBOK, INDONESIA, SEPTEMBER 1983



1. The food should be widely eaten by the population in the target area;
2. The daily intake of the food per person should not fluctuate much;
3. Addition of vitamin A should not cause any major modification in the organoleptic properties of the food;
4. The fortification of the food on an industrial scale must be economically feasible.

Vitamin A is generally incorporated in foods in the form of a retinyl ester (acetate or palmitate). Since it is sensitive to oxidation, which is hastened by heat, light and certain catalysts, techniques for incorporating it while inhibiting oxidation or delaying it through physical or chemical manipulations have been developed. Several vitamin A preparations have therefore been commercialized, each with its specific properties and uses for the fortification of food.

Milk products

The skimming of milk eliminates the fat-soluble vitamins contained in it, including vitamin A. The reasons for reincorporating vitamin A in liquid skimmed (defatted) milk are well known, as are the techniques. Vitamin A is sufficiently stable when liquid skimmed milk is correctly fortified. Vitamin A has also been successfully added to powdered milk and sweetened condensed milk.

All powdered skimmed milk donated by the United States, the European Common Market and the World food programme within their aid programmes for the developing countries is systematically fortified with vitamin A. The usual proportion is 5,000 IU per 100 g of dry powder.

Grain foods

The development, in recent years, of stable, dry forms of vitamin A, in various-sized particles, has greatly broadened the range of possibilities for food fortification with vitamin A. It is now possible to fortify a number of products such as wheat flour and maize flour, which could not be fortified with liquid preparations of vitamin A. Wheat and maize flours constitute excellent vehicles for fortification with vitamin A, since they are extensively consumed throughout the world. Binding of vitamin A in tortillas, buns, chapattis and bread is satisfactory and may be excellent.

Sugar

In 1976, a project for the fortification of a vehicle with vitamin A, on a national scale, was launched in Guatemala. White table sugar was chosen, since it is used by the entire population, the daily intake per inhabitant hardly varies, and addition of the vitamin does not notably modify its organoleptic properties. The cost and nature of the operation make it economically feasible on an industrial scale. Dry, water-soluble vitamin A palmitate was used. Tests for chemical stability, biological efficiency, taste and acceptability were all positive.

This project was evaluated in 1979, and was found to have induced a highly significant rise in the serum vitamin A concentration (Figure 9) of the population (9). An increase was also noted in the vitamin A content of breast milk, as well as in the hepatic reserves of a great many individuals. In 1979 the cost of fortification was estimated at approximately $US 0.08 per person and per year.

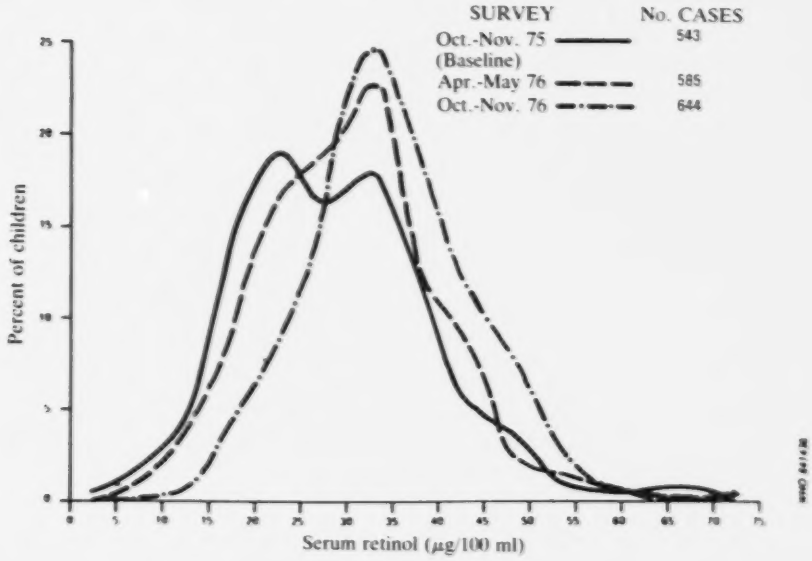
Another solution now being experimented is the fortification of monosodium glutamate, which is commonly used for flavouring in countries where rice is the staple food. Trials are under way in Indonesia and the Philippines. The results are quite encouraging, so far, with regard to both cost and efficiency.

IV - An overview of prophylaxis

Three main "services" - health, industrial (or agricultural) production of food and education - have been seen to contribute substantially to the prevention of xerophthalmia. All three should be put to work in many countries, and their activities should be coordinated, and directed toward similar objectives.

Fig. 9

EFFECT OF SUGAR FORTIFICATION WITH VITAMIN A ON THE DISTRIBUTION OF SERUM RETINOL CONCENTRATIONS OF RURAL PRESCHOOL CHILDREN 1975-1976
(after one year of fortification)



Health services

The health services should detect the presence of xerophthalmia and vitamin A deficiency within population groups and estimate their prevalence. They should be able to study the epidemiology of the deficiency in various ecological areas, and to set up a strategy for prevention. This means that they must dispose of workers trained in the diagnosis, treatment and prevention of xerophthalmia.

Xerophthalmia is usually most widespread in the poorest, most underprivileged social groups. Such families rarely have access to the existing health services, or make little use of them. A health service composed of numerous units working in the field and encouraging the use of auxiliary workers may therefore begin a better position to deal with this problem than a service based on a few urban hospitals and a personnel composed mainly of doctors. Auxiliary workers should be familiar with the diagnosis, treatment and prevention of xerophthalmia. A sufficient supply of vitamin A capsules (200,000 IU) should be constantly available in all health units, as well.

V - Horticulture and related activities

In most, if not all, of those parts of the world where xerophthalmia is present, the main source of dietary vitamin A (carotenoids) is plant foods rather than animal products. With the exception of red palm oil, it is certain vegetables (especially carrots and leafy, green vegetables) as well as fruits, which contain the most carotenoids. Some staple foods such as maize also furnish some beta-carotene. However, many horticultural products are only available at definite seasons, and this may influence prevalence, and result in the seasonal occurrence of vitamin A deficiency.

Few horticultural programmes have been launched in the framework of the xerophthalmia control programme so far, and an enormous amount of work remains to be done in this field.

Education programmes

There has never been any evaluation of the effectiveness of nutrition education programmes aimed at improving the consumption of vitamin A - rich foods. The importance of education specially aimed at at-risk families should be acknowledged, however. It should deal with the production and use of food products and, in some cases, with storage techniques as well. Families should be informed of the special dietary requirements of small children, and should know how to satisfy these, in practical terms.

CONCLUSION

Vitamin A deficiency and xerophthalmia are now known to be major public health problems in many developing countries, owing to the ocular damage and blindness caused by them, as well as their effects on morbidity and mortality of preschool children. The number of children affected and the geographic distribution of the problem are considerably greater than we imagined some years ago.

For this reason, the World Health Assembly sent out a warning, in May 1984, and recommended that the Organization undertake a vast programme, aimed at controlling and preventing vitamin A deficiency and xerophthalmia. Such an operation should be run in collaboration with all of the governments involved, other United Nations organizations such as the FAO and UNICEF, and a number of non-governmental organizations working toward the prevention of blindness. This programme was launched in 1985, and is presently developing rapidly. Many countries have expressed their desire to participate in it, and have requested technical assistance from WHO. One objective indication of the rapid development of the programme is the number of capsules of vitamin A distributed: it has practically tripled in the last three years.

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ANNEXE

THE ANATOMY OF THE HUMAN EYE

The visual apparatus is composed of a transmitter-receiver, the eyeball, inserted in a rigid frame: the socket or orbit, and protected by various envelopes: the adnexa.

1. THE ORBIT

The orbit, located at the joining of the skull bones and the face bones, has the shape of a quadrangular pyramid: its base, the orbital margin, is wide open in front, and its apex, to the rear, is in communication with the middle level of the brain by the optic foramen and the sphenoidal fissure, through which the various vascular and neural elements necessarily pass.

2. THE EYEBALL

The eyeball is an irregular, hollow sphere with a diameter of about 24 mm, closed by a shell and containing transparent humours.

1. The container

It is composed of three membranes, more or less closely joined. From within outward, they are:

a) A thick, fibrous, protective membrane composed of:

- *The sclerotic or sclera*, which constitutes the posterior 4/5ths. It is white, and opaque to light waves. At the posterior pole it is interrupted by the scleral canal containing the fibres of the optic nerve which are leaving the eyeball.
- *The cornea*, in front, is a transparent window inserted in the sclera and representing the first system for the convergence of light waves.
- At the joining-place of the sclera and the cornea, there is a transitional area of great anatomical and pathological importance: *the limbus corneae*.

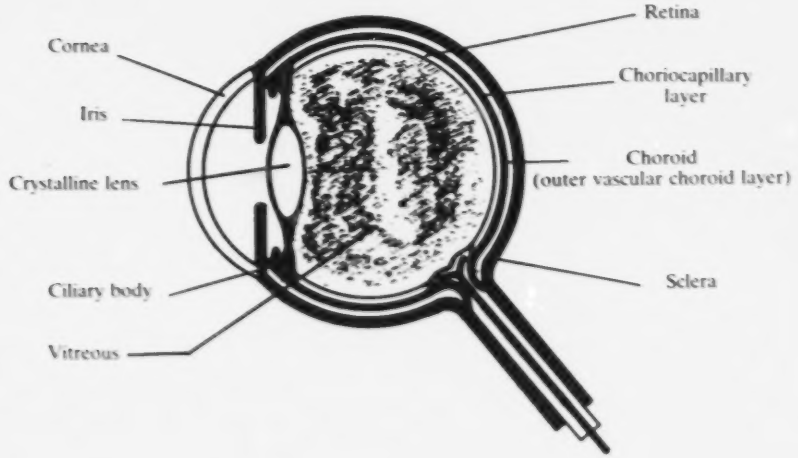
b) The uvea

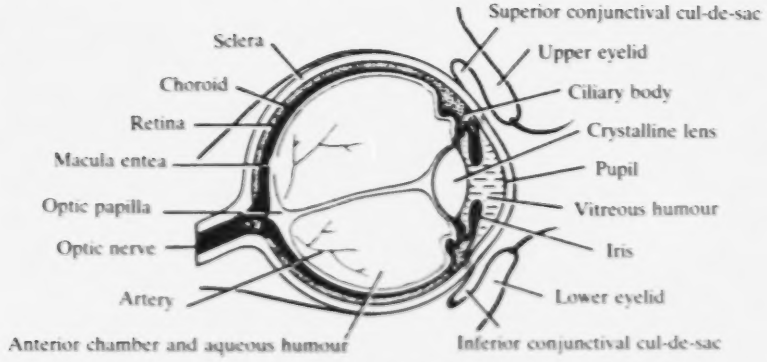
Its rich vascularization makes it the "hot plate" of the eyeball. It nourishes the underlying nerve cells, and is also highly pigmented and therefore absorbs the surplus light rays which strike the eyeball.

— Its posterior part or *choroid* lines the sclera.

— Its enlarged middle part is the *ciliary body*.

— Its anterior part is placed frontally and constitutes the *iris* which is a curtain surrounding the pupil. The iridal diaphragm is activated by a smooth muscle system. It varies the opening of the pupil, and thus truly doses the amount of light received.





CROSS-SECTION OF AN EYE

c) The retina

This is the sensory membrane. It is composed of several layers of cells which are excited by light, change it into nerve-impulses and transmit these to the fibres of the optic nerve.

The receiver role is played essentially by two types of highly differentiated cells: the cones and the rods. Accurate vision of shapes is more specifically commanded by the cones, which are more numerous in the macula lutea, the centre of acute vision, "the eye of the eye". The optic fibres converge towards the optic papilla which has no visual cells and constitutes the blind spot of the retina.

The optic nerve, which will be crossed with the nerve from the other eye in the chiasm, is born here.

The central retinal blood vessels enter through the papilla, and their many branches form a fine network, clearly visible when the fundus of the eye is examined.

2. The contents

a) The aqueous humour fills the anterior chamber, a space defined by the cornea in the front, the iris and the anterior face of the crystalline lens in the back. It is secreted by the ciliary body, flows into the anterior chamber and leaves the eyeball by complex efferent routes at the level of the limbus corneae.

b) The crystalline lens

This is a transparent, biconvex lens, located behind the iris. Within the optic system of the eye, it is mainly responsible for the focussing of light rays on the retina.

It constantly succeeds in doing so regardless of the distance of the focussed object, thanks to the reflex muscular mechanism of accommodation, which modifies the size of the lens, and therefore its power of convergence. It is held in place in the frontal plane by the fibres of the ciliary zonule of Zinn, which join the equator of the crystalline lens to the ciliary body.

c) The vitreous humour

This is a transparent jelly-like substance filling the space between the retina and the posterior side of the crystalline lens and which ensures the elasticity of the eyeball.

3. THE ADNEXA

The eyeball moves freely within its orbit, through the complex interaction of the oculomotor muscles, in a medium which serves as a shock-absorber. In front, the eyelid closes the orbit, protecting the eyeball.

1. The oculomotor muscles surround the eyeball, attach it to the orbital walls and provide its mobility.

2. The eyelids

Each eyelid is composed of a fibrocartilaginous structure lined with a muscular layer in front and a mucous membrane in the rear, the conjunctiva which is bent around the limbus.

3. The lachrymal apparatus

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